A CLINICAL STUDY OF INFANTILE HEMIPLEGIA.

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DR. CHARLES K. MILLS, during his term of service at the Pennsylvania Training School for Feeble-Minded Children, has permitted us to examine with him and to publish the following cases of infantile hemiplegia. We can not adequately express our appreciation of this and other acts of kindness on his part, and beg that he will accept this feeble acknowledgment.[1]

Few subjects have been more carefully studied than that of hemiplegia, and in the study of the infantile forms of paralysis American authors have distinguished themselves almost more than in any other branch of neurology.

It is, perhaps, well to state at once that in devoting our attention to infantile hemiplegia we may be exposed to a criticism similar to that bestowed by Sachs[2] upon the work of Freud and Rie. Infantile hemiplegia should not be separated from infantile diplegia or paraplegia; they are all three merely clinical manifestations of similar lesions of which the location alone is different, and a lesion which, in a given case, has caused hemiplegia might have caused diplegia had it been more extensive. Merely for the sake of convenience we have selected a certain group of cases and have attempted to study the clinical pictures presented. The time at our disposal was limited, and it was necessary to make some classification in order to use the rich material afforded by the Pennsylvania Training School for Feeble Minded Children.

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[1] The excellent photographs (numbers 2, 3, 4 and 5) have been taken under the supervision of Dr. Frank White. For photograph number 6 we are indebted to Dr. Robert Formad, and for number 7 to Mr. G. C. Bird, Jr. To Drs. Barr, Llewellyn, White and Morris we desire to express our appreciation of most valuable assistance.

[2] Sachs, Volkmann's Sammlung klin. Vorträge, 1–29, p. 438.

We have examined in detail thirty-three cases of hemiplegia from the Elwyn institution; twenty-three of these were girls, ten were boys, seventeen were cases of right, sixteen of left hemiplegia. A more equal division in regard to the side affected could not be desired.

In six cases facial paralysis in the distribution of the lower branch of the seventh nerve was observed; in three the naso labial fold on one side was less prominent. König[2] has confirmed the statements of Freud and Rie[3] in regard to the frequency of involvement of the muscles in infantile cerebral paralysis innervated by the seventh nerve. These authors have noticed some involvement of the facial muscles in twenty-five of their thirty-five cases.

We have not found the muscles supplied by the motor branch of the fifth affected. These are sometimes paralyzed in hemiplegia,as personal examination of cases in the adult has taught us.

In four of our patients the tongue was found paretic on the side of the hemiplegia, and in one there was marked unilateral atrophy.

The diminution in the size of the thorax is very common, but lessened expansion on the paralyzed side was not observed. In sixteen out of the twenty-eight cases which could be examined the thorax on the paralyzed side was smaller.

In some patients the difference was as great as an inch and a quarter, or even an inch and three-quarters. Great care was observed in order to avoid mistake, and where the difference was very little it was not recorded. The thorax does not appear to be smaller when the upper extremity is well formed, but in a few cases we have found the upper extremity much affected, and yet the thorax was of normal development.

Impairment of the intellectual faculties, to a marked degree, was noticed in thirteen cases. In some of the other patients the intellect was not of a high grade.

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[2] König, Neurologisches Centralblatt, 1895, p. 797.

[3] Freud and Rie, Klinische Studie über die halbseitige Cerebralähmung der Kinder.

Sachs[4] states, in his recent work, that he has found idiocy in thirteen per cent. of the subjects of hemiplegia. This seems to us, judging from our cases, which may, perhaps, offer an unfair standard, rather an under estimate. The degree of idiocy does not seem to bear any constant relation to the degree of the hemiplegia.

In none of these cases were the muscles of the neck involved, nor are we aware that this involvement has ever been observed. The only explanation is that the innervation of the muscles of each side of the neck from both hemispheres of the brain is so perfect that an unilateral cerebral lesion can cause no paralysis of these muscles. The same is true of the muscles of the larynx. This is an opinion held by Krause, Semon and Horsley, and Broeckaert and recently supported by Simerka.[5]

In progressive muscular atrophy the upper part of the trapezius muscle commonly survives after the lower portion of it has been destroyed. There may be an explanation for this as given above. The ganglion cells in the anterior horns of the spinal cord which supply this part may have a more perfect double innervation, and through this stimulus preserve their vitality longer than do those which supply the lower part of the muscle. Marinesco and Goldscheider have advanced the theory that motor cells preserve their vitality only so long as they receive normal impulses.

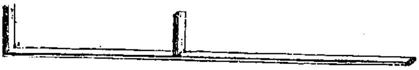

Fig I.

The muscles of the two sides of the neck move in more perfect unison than do those of the back and shoulder girdle, and the upper part of the trapezius is constantly employed in maintaining the erect position of the head.

We have not found aphasia, even in right hemiplegia, in any case in which the impairment of the intellect was not of high degree, except in one case where the patient was both deaf and dumb. It is well known that aphasia, either sensory or motor, occurring in childhood, usually completely disappears. Wernicke's case, quoted in the recent excellent thesis of Miralliè[6], tends to confirm the view that the speech centres of the right hemisphere become active when the normal centres are destroyed. He reported the case of an adult who was aphasic and paralyzed on the right side. Speech was regained. A second attack of apoplexy reproduced the aphasia, and this time it was associated with left hemiplegia. At the autopsy an old lesion occupied the centre of Broca in the left hemisphere, and a more recent one was found in the foot of the right third frontal convolution. Oppenheim[7] holds the opinion that the speech centres of the right side of the brain, even in right-handed persons, are partially developed. A most interesting statement is made by Sachs.[8] According to him, the proportion of aphasia in children with left hemiplegia is relatively large, and the explanation which he offers is that at this period of life the differentiation in the hemispheres is not as perfect as in adult life.

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[4] Sachs, Nervous Diseases of Children, p. 538.
[5] Simerka Revue Neurologique, June 15, 1896.

Shortening and general diminution in size of the paralyzed limbs was found in thirty-one of the cases, and, as has been recorded by other writers, this was always greater at the peripheral extremities. We will not attempt to give figures in regard to the length of the limbs. Féré[9] has recently studied this subject in a number of cases.

For the determination of the length of a limb Dr. Llewellyn suggests the use of the instrument employed by shoemakers in measuring the length of the human foot. It may be employed with advantage (see Fig. I).

In eight of these cases of right hemiplegia, where the intelligence was of sufficient grade to permit a test of the writing to be made, it was found that spontaneous writing was possible in five cases; one patient could print a few words, another could copy script as script, another could not write at all. Imperfect education may have been the cause in these three cases.

Sensation was not altered in any of our cases, nor was it in any of those reported by Osler.[10] Oulmont has, however, observed disturbance of sensation, and Raymond has seen hemianæsthesia once (quoted by Marie).

Epileptiform convulsions have been observed in twenty-two cases. In five more convulsions occurred in infancy. Sachs and Peterson believe that forty-four per cent. of all cases of infantile paralysis develop epilepsy, and Sachs thinks that a very fair proportion of cases of ordinary epilepsy are developed in connection with infantile palsies, which may not be recognized. The percentage in our cases is much greater than forty-four. In fully ninety-five per cent. the initial lesion of infantile palsy is in or upon the cortex, according to Sachs,[11] and is usually hemorrhage, thrombosis, or embolism. This causes a development of secondary sclerosis throughout the cortex with rather surprising rapidity. The frequency of the cortical location explains the frequency of the convulsions. According to Marie[12] the epilepsy of infantile hemiplegia is peculiar; there is an absence of foam and blood from the mouth, as the tongue is rarely bitten, very seldom are there involuntary evacuations, the attacks end abruptly, there is no period of stertor or coma, and when convulsive movements have ceased consciousness returns, and the child resumes its play as if nothing had happened. After the epilepsy has lasted for a long time these differences may not be noticed.

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[6] Miralliè, De l'Aphasie Sensorielle.

[7] Oppenheim, Die Geschwülste des Gehirns, p. 85.

[8] Sachs, Nervous Diseases of Children, p. 532.

[9] Féré, Revue de Médecine, No. 2, 1896.

[10] Osler, The Cerebral Palsies of Children.

Gowers[13] states that ultimately the fits may be preceded by an aura identical with some of the common warnings of idiopathic epilepsy. We have found that in at least six of our patients the approaching attack could always be foretold by an aura. One patient has paræsthesia of the throat, and pain in the region of the heart; another has spasmodic movements of the body; another perspires freely on the head and shoulders, and has a rush of blood to the head; three have peculiar sensations located in the stomach. Some of the other patients are of too feeble intellect to make any statement in this respect.

The etiology of these cases is very difficult to obtain. Carefully worded blanks are sent to the relatives of the children admitted to the institution, but they are often imperfectly filled out. In many cases convulsions occurring in the first few months after birth are given as the cause. It seems to us these should in general be regarded as the first manifestations of a previous lesion, and in this we incline toward the opinion of Freud[14], Dercum,[15] etc. Dercum believes also that permanent changes in the nutrition of the cortex may occur in some cases from prolonged spasms. Osler and Sachs are much in favor of the view that convulsions may be the cause of hemiplegia, and their arguments are forcible. In one of our cases we find scarlet fever given as a cause, in another "a congestive chill, followed by apoplexy and unconsciousness for four days;" in another "brain fever," in another "catarrhal fever," in another small-pox. Lovett could in one case only associate the beginning of the paralysis with an acute infectious disease, and that was diphtheria. Imogene Bassette[16] has pulished a number of cases of paralysis following nearly all the infectious diseases of childhood, although in many of her cases neuritis is prominent. Jendrassik and Marie[17] also speak of infectious diseases as a cause of hemiplegia. Cotard recognized this factor at a very early date. Osler has noted a connection with infectious diseases in sixteen of his cases. (l. c.) Freud and Rie[18] were able to refer five of their thirty-five cases of infantile hemiplegia to this origin. Moraga[19] describes hemiplegia at the age of twenty-two months after measles, and Oppenheim[20] at the age of eight years after whooping cough. Numerous other cases are found in the literature, but the tendency, especially among French writers (Sachs), has been to ascribe too many cases to infectious diseases.

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[11] Sachs, The New York Medical Journal, Feb. 1892

[12] Marie, Dictionnaire Encyclopêdique des Sciences Médicales, 1888.

[13] Gowers, Diseases of the Nervous System, Vol II. p. 459 (English edition).

Marie[21] thinks that hemiplegia beginning after the age of nine should not be called infantile. In forty cases of infantile hemiplegia examined by Sachs[22] the causes were atrophy, sclerosis and cysts; in two cases porencephaly was found, in twenty three hemorrhage, in seven embolism, in five thrombosis, in one agenesis, and in another tubercle. Intracerebral hemorrhage in the young is more common than is generally supposed (Sachs). Hereditary syphilis does not seem to be of much importance (Sachs, Osler). The remarkable case of aneurism of a branch of the anterior cerebral artery in a boy of six years, recorded by Osler[23], is well known. Dejerine[24] reports three cases of infantile hemiplegia with autopsy. In two of these the lesions were due to hemorrhage; the third was probably due to ischæmia. One case occurred in convalescence from scarlet fever. Starr[25] gives his opinion very firmly in favor of cerebral hemorrhage as the cause of these birth palsies, and Osler favors embolism (l. c.) Schultze[26] has recently reminded us that hemorrhage during birth may occur in the spinal cord, and not necessarily in the brain. The frequency of this is doubtless considerably less than that of cerebral hemorrhage. Pfeiffer[27] expresses this view. He examined five cases; in four of these the forceps had been employed, in one version had been practiced. Only once did he find macroscopically the signs of extensive hemorrhage in the cord, and he concluded that hæmatomyelia from dystocia should not be regarded as com-

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[14] Freud, Zur Kenntniss der cerebralen Diplegien des Kindesalters, p. 60.

[15] Dercum, Nervous Diseases, p. 509.

[16] Imogene Bassette, Journal of Nervous and Mental Disease, 1892.

[17] Jendrassik and Marie, Archives de Physiologie, 1885, p. 51.

[18] Freud and Rie, Klinische Studie über die halbseitige Cerebrallähmung der Kinder.

[19] Moraga, abstract in Neurologisches Centralblatt, No. 11, 1896.

[20] Oppenheim, Deutsche Med. Wochenschrift, 1896, No. 17.

[21] Marie, Dictionnaire Encyclopédique des Sciences Médicales, 1888.

[22] Sachs, Sammlung klinischer Vorträge, Inner. Med., 1-29, p. 470.

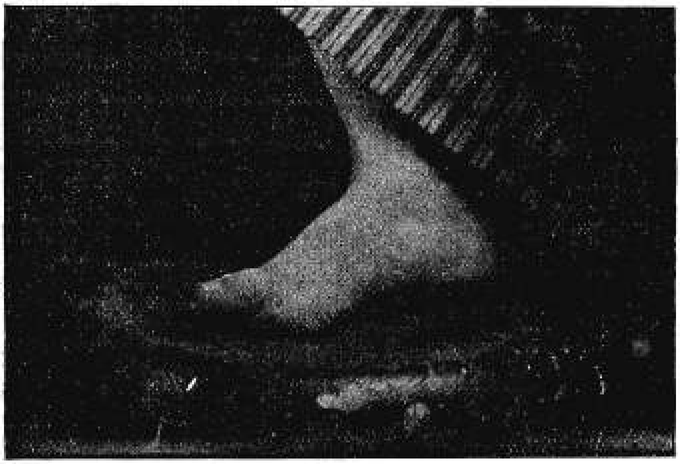

FIG. II.—Hyperextension of the big toe in infantile hemiplegia.

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[23] Osler, Canada Medical and Surgical Journal, 1886 (quoted by Sachs).

[24] Dejerine, Archives de Physiologie, 1891, p. 661.

[25] Starr, Medical Record, 1892

[26] Schultze, "Deutsche Zeitschrift für Nervenheilkunde, VIII., Nos. 1 and 2.

[27] Pfeiffer, Centralblatt für Allgemeine Pathologie u. Pathologische Anatomie, Sept., 1896.

mon as the first investigations seemed to indicate. Nevertheless, one out of five cases seems to us a large percentage, and further examination will probably show that it is too great. Peterson,[28] referring to the fact that Herbert R. Spencer, in a hundred and thirty itsllborn children, found hemorrhages into the spinal canal and cord in thirty cases, says he cannot but believe that some, though a very small percentage, of the cases of paraplegia especially, and perhaps diplegia, may be due to cord lesions at birth after all, and not to cerebral lesions.

In some of our cases the hemiplegia was first noticed after convulsions at the age of four, five or six years. It is not improbable that a lesion may have occurred during birth, or within the intrauterine period, and that the sclerosis may have taken a few years to reach a stage sufficient to cause convulsions and hemiplegia. The case we present in this paper, with a photograph of the brain (Fig. VI.), is in evidence of the long duration of sclerosis without epilepsy. In another case, which was congenital, the epilepsy first developed at the age of twelve.

We hardly think it worth while to note the statements in regard to difficult labor, inasmuch as we are unable to obtain reliable answers from the parents. These blanks have not been filled out by the attending physicians, and many women are inclined to regard a normal labor as difficult. We do not mean that this question is of little importance in infantile paralysis, for Little, Gowers, and others in England; Sarah McNutt, Sinkler, etc., in this country, have taught us differently, and yet Lovett says: "It seems reasonable to conclude that the influence of difficult labor in producing cerebral paralysis must have been greatly overestimated." He also quotes Langdon Down as saying: "I found that among the great number of feeble-minded children about whom I could get thoroughly reliable accounts, in only three per cent. had the forceps or any other instrument been used."

It is also not worth while to enter into a discussion of Strümpell's theory in regard to acute infantile encephalitis, as we have no facts to bring forward for or against this view.

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[28] Peterson, An American Text-Book of the Diseases of Children. Edited by Louis Starr. p. 657.

The French authors especially have made very prominent the peculiar condition of the foot in Friedeich's disease, of which one of the chief features is the hyperextension of the big toe, and to a less degree of the others. The first phalanx is over-extended, while the second is flexed. This hyperextension may be excessive. The position of equino-varus is usually present, but is not essential, according to Brissaud[29] Two excellent pictures of the condition are given by this author. Marie[30] also describes this alteration of the foot, and regards it as a relatively early sign of Friedreich's disease. In certain families, in which several members are afflicted with this malady, the hyperextension of the big toe indicates the beginning of the process. Auscher[31] also presents a picture of the deformity in a case of Friedreich's disease. Higier[32] has reported four remarkable cases, which he considers atypical forms of cerebral diplegia. In all four

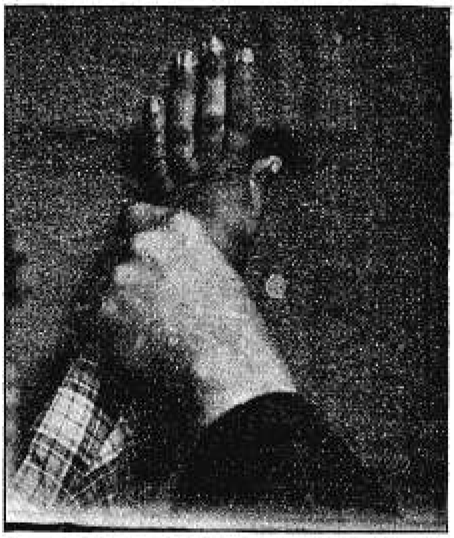

Fig. III.—Ulnar Oligodactyly.

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[29] Brissaud, Leçons, sur les Maladies Nerveuses, pp. 57, 58.
[30] Marie, Leçons, sur les Maladies de la Moelle, p. 391.
[31] Auscher, Archives de Physiologie, 1893, p. 342.
[32] Higier, Deutsche Zeitschrift für Nervenheilkunde, Vol. 9, 1896.

this hyperextension of the big toe is well represented in the pictures. Two of our cases of hemiplegia present this deformity (see Fig. II.). We are not aware that this has been mentioned instudies on hemiplegia. We would imagine from the picture that it was present in Sachs'[33] case of congenital diplegia known as the "Frog Girl." This hyperextension, however, can not be considered as pathognomonic of Friedreich's disease.

Féré[34] has devoted much attention to the deformities in infantile hemiplegia. He has described a condition which he calls ulnar oligodactyly ("oligodactylie cubitale"), which is seen chiefly in the ring finger. The little finger often takes part in the anomaly. Occasionally, in normal persons, the ring finger is shorter than the index, but in forty two cases of infantile hemiplegia with atrophy of the hand Féré found this condition thirty-six times. We have observed this shortness of the ring finger in nine cases (see Fig. III.). In four cases athetoid movements have been noted.

Occasionally the cerebral form of hemiplegia may be difficult to distinguish from the spinal. The following case is an example of hypotonia in cerebral hemiplegia.

Case No. 1.—The paralysis is in the right upper and lower limbs. Voluntary movement at the right shoulder and elbow is restricted and awkward. In the fingers of the right hand the voluntary movement is very imperfect, and yet the right arm can be raised passively above the head as high as the non-paralyzed limb, the forearm can be flexed passively with great ease. There is no resistance in the right shoulder and elbow to passive movement. There is a slight tendency to flexor contracture at the wrist, which can be easily and fully overcome. There is also a slight tendency to flexor contracture in the fingers. The right upper extremity is shorter than the left, and the difference is very noticeable in the hand. The musculature of the entire limb is less developed than that of the left. In the right lower limb passive movement reveals also a condition of moderate hypotonia. The foot is in the position of slight talipes equinus, but can be easily extended, and in walking the entire foot is placed upon the ground. Active movement is free at all joints of the lower limb, but the right knee is raised a little higher than is normal in walking, as the toes of the right foot have a tendency to cling to the ground. The patient has on this side a very slight degree of the "stepper gait," as it is called by Charcot. The entire right lower limb is shorter than the left, but the difference is in the leg and foot; the thigh is about normal in length. The musculature of the entire limb is smaller en masse. The patella reflex is present on both sides, but is not exaggerated. The reflexes of the right upper limb are not increased.

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[33] Sachs, Nervous Diseases of Children, p. 531.
[34] Féré, Revue de Mêdecine, Feb., 1896, p. 115.

On account of this condition of hypotonia, which is very perceptible, though not excessive, and seems to be more than imperfect development of the muscles would account for, we have hesitated at first in excluding poliomyelitis. The preservation of the reflexes, the slight tendency to contracture and the diminution in size of the hand and foot, parts which are supposed to be more exclusively under the control of one side of the brain than the rest of the limbs, as the generally accepted theory teaches, and the very feeble mental condition, make the case undoubtedly of cerebral origin. We note also the considerable diminution in the right thorax, amounting to an inch and a quarter, although the right arm and thigh are not much smaller, the differences being chiefly in the forearm and hand leg and foot.

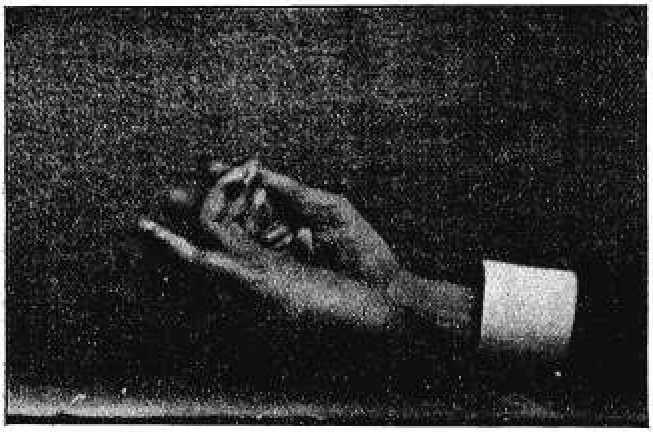

FIG. IV.—Hypotonia of fingers in the paralyzed hand.

Sachs and Peterson[35] report two cases of cerebral palsy in which the element of spasticity was entirely wanting, although the cases were undoubtedly of cerebral origin, the proof of which was furnished by the post mortem examination in one of the two cases. Osler also reports three cases in which the arm was flaccid (l. c.) Usually, as in the case we report, there are sufficient signs to make the cerebral nature of the affection evident, even where spasticity is absent. In another paper Sachs[36] makes the following statements: "I can safely say that of the two hundred and five cases we have seen, doubt as to the spinal or cerebral origin was entertained in but a single instance." "Since the foregoing was written, I have seen one case, and the only one, in which there was evidence of a spinal and a cerebral lesion."

Case No. 3 is an example of retardation of growth in the extremities of the paralyzed limbs, which we have noticed in so many of these cases. The lower limbs are about the same length, and yet the foot of the paralyzed side is much smaller than that of the other side. In the upper limbs the difference is slight in the length of the two forearms, but in the hands this is much greater. There seems to be no explanation for this if we reject the theory that the extremities of the limbs are more exclusively innervated from one side of the brain. It is well known that motion is usually more impaired in hemiplegia at the extremity of the limb.

We may, if we wish, believe that the degeneration of the fibres in the crossed pyramidal tract of the "sound" side in a case of hemiplegia begins at the motor decussation, and Rothmann[37] has recently explained this as the result of pressure on the fibres coming from the normal pyramid by the degenerating fibres from the affected pyramid. In the early stages of degeneration there is swelling of the fibres and pressure is exerted. This is a little different from the theory of Hallopeau.[38] According to the view of this writer, the degeneration of the pyramidal tract of the "sound" side begins also at the decussation, but the cause is transmitted inflammation from the fibres of the degenerated pyramid to those of the sound. This theory of the origin of the degeneration of the fibres of the crossed pyramidal tract of the "sound" side at the decussation does not explain such a condition as is presented in these cases. We can not understand why the extremities of the limbs are so often more involved on the paralyzed side unless we believe that, while those portions of the limbs nearest the trunk have a bilateral, the hand and foot have a more exclusively unilateral cerebral innervation. If the degeneration of the crossed pyramidal tract, which has been found on the "sound" side, does not represent fibres which come from the cerebral hemisphere on the same side as the lesion, then we have little or no anatomical proof that such fibres really exist, and the innervation of each limb from both sides of the cerebrum must remain merely as a theory. Rothmann, however, does not stand on the side of the majority of investigators. Dejerine and Thomas[39] state they have seen degenerated fibres passing from one pyramid to the crossed pyramidal tract of the same side. Rothmann describes similar fibres, but be-

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[35] Sachs and Peterson, Journal of Nervous and Mental Disease, 1890.

[36] Sachs, New York Medical Journal, 1891, p. 503.

[37] Rothmann, Neurologisches Centralblatt, Nos. 11 and 12, 1896.

[38] Hallopeau, Archives Generales de Mêdecine, Vol. 11, 1871. (Quoted by Rothmann.)

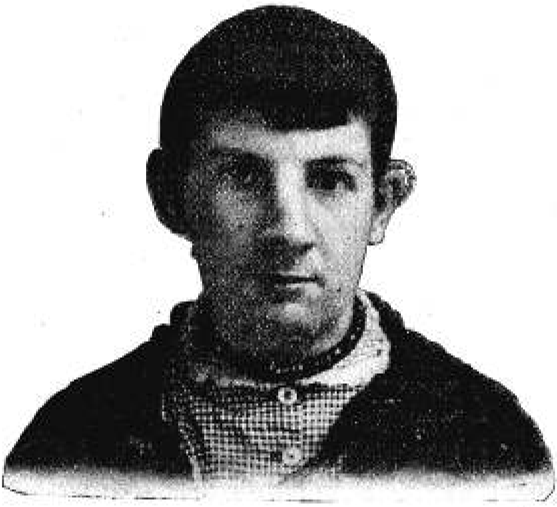

FIG. V.—Unilateral arrest of development of the face in infantile hemiplegia.

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[39] Dejerine and Thomas, Archives de Physiologie, April, 1896,

lieves that their course excludes the possibility of their origin in the pyramid of the same side.

It is well known that the foot can be trained to make almost all the movements of the hand. We can recall an artist in the art gallery of Antwerp who, being deprived of both arms, made an excellent copy of one of the paintings by means of his feet—mixed his colors and picked up his brushes and palette by means of his toes alone. It seems easy of comprehension that for these highly specialized movements of hand and foot special cells on one side of the brain should be set apart, while for those movements of both sides of the body which usually occur together both cerebral hemispheres should be employed. How else shall we explain associated movements in paralyzed limbs?

In examining spinal cords from cases of hemiplegia by the method of Marchi we have found several times both crossed pyramidal tracts degenerated. This is by no means a recent discovery. It must be remembered that there is no truly sound side in hemiplegia.

The fingers and thumb in this case (No. 3) are in flexor contracture. In many of these cases of contracture of the wrist joint the fingers are in extension.

The view of Rothmann that the degeneration of the crossed pyramidal tract on the so-called sound side begins at the motor decussation explains the increased reflex of the "sound" side as well as does the theory that fibres extend from the cortex to the lateral column of the same side of the cord.

Cases Nos. 4 and 5 are examples of the abnormal flaccidity (hypotonia) of the fingers on the paralyzed side, seen in at least six of these children. The fingers can be bent backwards almost at a right angle (see Fig. IV.). We have noticed this flaccidity of the fingers even when the wrist was in flexor contracture.

In case 5 the upper limb on the paralyzed side is shorter than on the "sound" side, and the thorax is three-quarters of an inch smaller. There is impairment of motion at the shoulder but none at the hip, as is seen in many of these cases. This indicates, probably, that the two sides of the brain are concerned in the movements of the hip joint more than in those of the shoulder. Flexion of the fingers is much better than extension, as is usually the case, and abduction and adduction of the fingers are most interfered with.

Case No. 6 presents latent contracture of the wrist joint on the paralyzed side; any manipulation of this joint causes increased flexion of the hand. If the hand is allowed to rest quietly on the examiner's knee it remains partially extended It is a question if it would be wise in a case like this to resort to passive movement, as permanent contracture might develop. There are athetoid movements in the paralyzed hand.

Exaggeration of the reflexes may remain after motor power has been almost fully restored, as is seen in case No. 7. The patella reflex on the paretic side is exaggerated, but the gait is almost normal. This patient was born in normal labor. At the age of two she had a convulsion, which was followed by loss of sight, hearing and speech. Aphasia has entirely disappeared. This was a case of *left* hemiplegia. There is an uncertain history of brain disease when the child was eight months of age, which was supposed to have been the cause of the hemiplegia.

Case No. 13 has a very interesting feature. The patient has left hemiplegia, with paralysis of the muscles innervated by the lower branch of the seventh nerve, and the left side of the face is smaller than the right (see Fig. V.). It is true Féré[40] remarks that the face usually participates in the hemiatrophy of infantile hemiplegia, and is more or less asymmetrical. This is a somewhat surprising statement. We have only found one case of arrested development in which the face was involved.

Case No. 16, contrary to the customary condition, presents greater contracture at the elbow joint than at any part of the paralyzed upper limb.

Case No. 18 is one of the most interesting we have examined, and of practical importance. Wiener[41] has recently published a case of progressive muscular dystrophy which was considerably improved by methodic gymnastics. Zabludowski[42] has reported a case of Friedreich's ataxia which was much benefited by massage employed for four weeks. Nature, by means of athetosis teaches us how continued exercise of the paretic limbs develops the musculature. Contracture and athetosis seldom are present together in severe form. Sachs[43] recommends massage and electrical treatment in every case of cerebral paralysis, to prevent contracture. From this statement it would seem that there is no fear of developing the latent tendency to contracture, what the French call the "imminence" or "opportunité de contracture." Our case No. 18 employed such exercise of her own accord. These cases are encouraging in view of the hopelessness usually felt toward the possibility of improvement in such conditions. The patient, A. K., is very intelligent for her station in life. The left hemiplegia is supposed to have been congenital. The patella reflex on the two sides is equally increased, and ankle clonus is present on both sides. Reflexes of both upper extremities are exaggerated, though more on the paralyzed side. The patient can raise her left arm at the shoulder almost as high, and can draw it backward almost as far as the right. She can almost fully extend the left forearm, but cannot extend the hand beyond the plane of the forearm. She has considerable power of adduction and abduction in the fingers, and these movements we have found in all the other cases the most interfered with. She can flex the fingers, but has little power of flexion at the wrist, and opposition of thumb with fingers is very imperfect. The left upper limb is much shorter than the right, and the difference is more noticeable in the forearm and hand. The musculature of the left upper limb is somewhat less developed than that of the right, but the difference is not as great as is usually found in these cases, probably on account of the exercise of the muscles. The left hand and fingers are very much smaller, The grasp of the left hand is rather feeble. In the lower extremities the thighs cannot be flexed on the abdomen as much as in most of these cases, although the right can be flexed a little more than the left. This in connection with the equal exaggeration of the patella reflex on the two sides would seem to indicate that in this case the pyramidal tract on either side supplies the lower limbs more nearly equally. There is no power of flexion or extension at the left ankle joint; the toes of the left foot cannot be moved. The left lower limb is much shorter than the right, and the difference is greater below the knee. The musculature is also less developed. In walking the toes of the left foot touch the ground, and the heel is held elevated. The left side of the thorax is three-quarters of an inch smaller than the right.

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[40] Féré, Les Épilepsies et les Épileptiques, p. 31, also Revue de Médecine, No. 2, 1896.

[41] Wiener, Neurologisches' Centralblatt, May. 1896. The American Journal of the Medical Sciences, Oct., 1896.

[42] Zabludowski, Berliner klinische Wochenschrift, No. 34, 1896.

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[43] Sachs, Volkmann's Sammlung klinischer Vorträge.

In view of the exaggeration of the reflexes, passive movements might have been thought unadvisable, but the result has not justified such a fear. There is contracture only at the ankle and wrist. There are cases of infantile hemiplegia which improve spontaneously, and the question might arise if this is not one of these cases. The exaggeration of the patella reflex, the decided ankle clonus, and the increase of the reflexes in the upper limbs on both sides, the loss of power at the wrist and ankle, the retarded growth of both left limbs and of the left side of the thorax, make it doubtful if improvement would have been so great if means had not been used. The patient, who is intelligent and can read and write, informs us that up to the age of nine years the power in the left upper limb was much less than at present. At this period of life she began systematically to use the left upper limb in order "to see how much she could carry." She would pick up a pail of water and carry it in her left hand, and in this way power became gradually greater in the arm. This was her own idea. She would raise

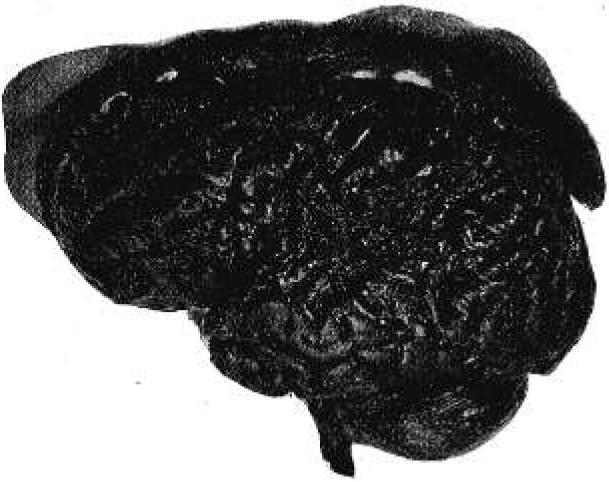

FIG. VI.—Sclerosis in the distribution of the Sylvian artery. From a case of infantile hemiplegia.

the left arm at the shoulder with the right hand, and would forcibly extend the left hand.

An interesting fact stated in the history is that the mother of this patient was also paralyzed in the left side. While we do not know that the mother was paralyzed previous to the birth of this child, we learn that she lived but fourteen months after the child was born. It looks very much as though the condition were hereditary in the daughter. Such cases have been recorded (Oppenheim).

Case No. 17 has a peculiar condition of the hand on the paralyzed side. The contracture at the wrist is extreme, the hand is so much flexed that it almost touches the front of the forearm. the fingers are also flexed, and the hand can only be extended passively far enough to form a right angle with the forearm. According to the statement of the patient, who is intelligent, and to the observations of those about him, every morning the hand is less contracted, and is only slightly flexed at the wrist, the fingers are then extended, and he has some power of movement. This condition lasts only a short time, and very soon the hand and fingers are again extremely flexed. The increased contracture as the day passes is probably due to summation of irritation brought about by constant demands on the reflex action. We can recall in the clinic of Professor Dejerine, at the Salpêtrière,a patient with myelitis,in whom the lower limbs showed this alternate condition of flexion and extension.

Case No. 30 exhibits contracture in both legs and one arm. The muscles of the left shoulder are much contracted and offer great resistance to passive movements in all directions; the forearm can only be extended passively to an angle of about 115° with the arm, the hand forms a right angle with the forearm, and cannot be moved by the patient in any direction. There is slight power of flexion in the fingers. The fingers can be moved passively in all directions.

Both lower extremities are equally rigid, abduction is lessened, flexion of the right thigh on the pelvis and of the right leg at the knee is not possible beyond a right angle. Active movement in the left lower limb is only a little less than in the right. The two legs are of equal length. The entire left upper extremity is much smaller than the right The two sides of the thorax are exactly equal.

In walking the left limb is dragged from the hip and the knee and ankle are held rigid. The right knee is kept partly flexed. The patella reflex is exaggerated on both sides, but more on the left Epileptic attacks occur at irregular intervals. Nothing is known of the early history of the patient. This condition may be due entirely to an unilateral lesion, or it may be that the lesion has extended over the superior part of the hemispheres and involved the leg centre of the opposite side.

Case No. 31 has a very interesting tremor on the paralyzed side. There are occasional involuntary twitchings of the left side of the jaw. The paralyzed arm is in a state of constant tremor, which is most noticeable in the hand. The same condition is observed in the lower limb. At times the tremor may be called fascicular, again it is so strong that the leg moves considerably. These post-hemiplegic movements are well known since Weir Mitchell long ago called attention to them.

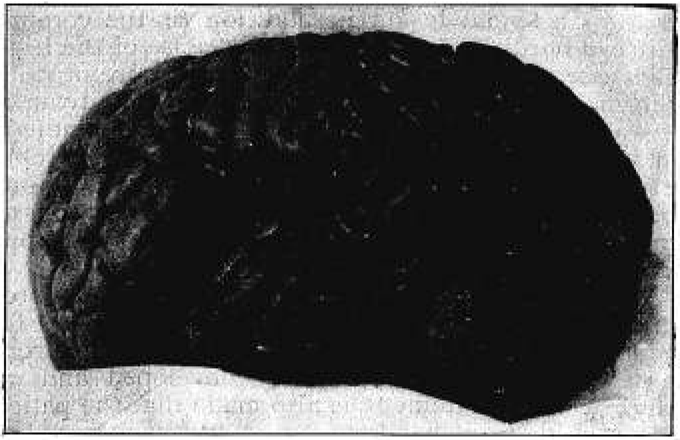

FIG. VII.—Porencephaly from a case of infantile hemiplegia.

We present a photograph (see Fig. VI.), kindly taken for us by Dr. Robert Formad, of a very interesting brain. We do not enter into a detailed description, as we are now studying the case by means of serial microscopic sections.

The patient was paralyzed on the right side "from a fright" in early infancy. Epilepsy did not develop until the age of ten. It is surprising that convulsions were not observed sooner, in view of the extensive sclerosis. The boy's vocabulary was very limited. It will be noticed that Broca's region was involved. Impairment of the mental faculties was present. Until the age of thirteen the boy's mother states that he was able to walk. At this period progression became impossible. As the right hemisphere presented no lesion at the autopsy, it is fair to assume that the weakness of the left lower limb was due to the sclerosis in the left hemisphere, and the case is in evidence of bilateral innervation from each hemisphere. The fact that the sclerotic area was limited to the distribution of the middle cerebral artery makes the theory of extensive progression of the process doubtful. Unfortunately we have not been able to examine the spinal cord, but there is no reason to suppose that there was a spinal lesion in addition to the cerebral. There can hardly be a doubt that the cause of the lesion in this case was an embolus or thrombus, more probably the former. Only the area nourished by the Sylvian artery was sclerotic. The upper part of the Rolandic region, which receives branches from the anterior cerebral artery, almost all the frontal, the whole of the occipital lobe, and the second and third temporal convolutions are normal. These parts do not receive their blood supply from the middle cerebral artery.

The clinical history of the interesting case of poren cephaly we present (see Fig. VII.) is very meagre. The patient's mental condition was of low grade. The right side of the body was imperfectly developed and was paralyzed. The statement is also made that the patient never used his right hand.

It will be noticed that the cavity involves Broca's area, and yet this boy is said to have "sung sweetly." He could not, therefore, have been aphasic, at least in singing.

More fibres were cut or remained undeveloped than a superficial examination would give reason to suspect, for we find by a microscopic study of the oblongata that the left pyramid is absent while the right is unusually large. The gyri do not end abruptly at the borders of the cavity, but with the pia pass into its depths. Later we will probably have something more to say of this interesting specimen.